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Neurological Sequelæ of Decompression in Supersonic Transport Aircraft

The forthcoming generation of supersonic transports will operate at altitudes far in excess of present-day subsonic aircraft. Altitudes around 70 000 ft may be used eventually, but immediate developments concern the range between 55 000 and 60 000 ft. The effect of a small structural failure must be carefully evaluated because, although emergency measures such as increased flow of air into the cabin and the descent of the aircraft will reduce the maximum altitude reached within the cabin, the occupants could be exposed to a prolonged period of hypoxia.

Early studies on the effects of small circular defects in the cabin were based on models and showed that a relatively small increase in the size of the defect led to a very considerable increase in the maximum cabin altitude reached and the duration of the exposure to high altitudes. With an 8 inch (20 cm) diameter defect the duration of exposure to altitudes above 45 000 ft would be such that, even if the occupants were breathing oxygen, they would suffer severe hypoxia which might well prove to be fatal. After a 6 inch (15 cm) diameter defect the cabin altitude would not exceed 45 000 ft but an altitude of 25 000 ft would be reached in about a minute after decompression. During this interval passengers breathing air would remain conscious and would be able to don oxygen masks. If the defect was equivalent to a 4 inch (10 cm) diameter hole the fall in cabin pressure would be more limited and it is likely that the maximum altitude reached of 30 000 ft would produce only transient unconsciousness in healthy passengers, even if no oxygen was available.

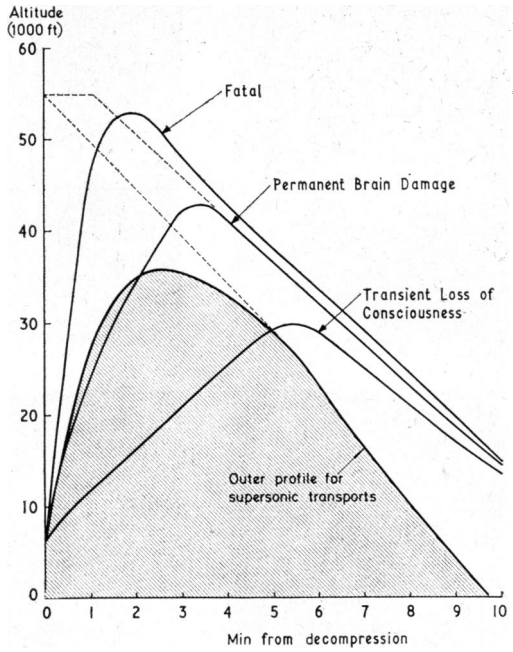

Fig 1 *Decompression profiles*

These considerations were based on the use of emergency oxygen, but observations were necessary to establish the effects of the decompressions in unprotected individuals. The studies were carried out in baboons (*Papio cynocephalus*) and it was evident that behavioural and neurological disturbances could result from such prolonged periods of hypoxia (Nicholson & Ernsting 1967). Three profiles were investigated which simulated decompressions arising from 4 inch, 6 inch and 8 inch diameter defects (Fig 1). The 4 inch diameter profile had a maximum altitude of 30 000 ft with a time of 3 minutes above 25 000 ft while the 8 inch diameter profile had a maximum altitude of 53 000 ft with a time of 1 minute 25 seconds above 50 000 ft. The former decompression led to a transient loss of consciousness with complete recovery while the latter was fatal. The decompression profile which simulated a 6 inch diameter defect had a maximum altitude of 42 500 ft and a time of 1 minute 30 seconds above 40 000 ft. This decompression was compatible

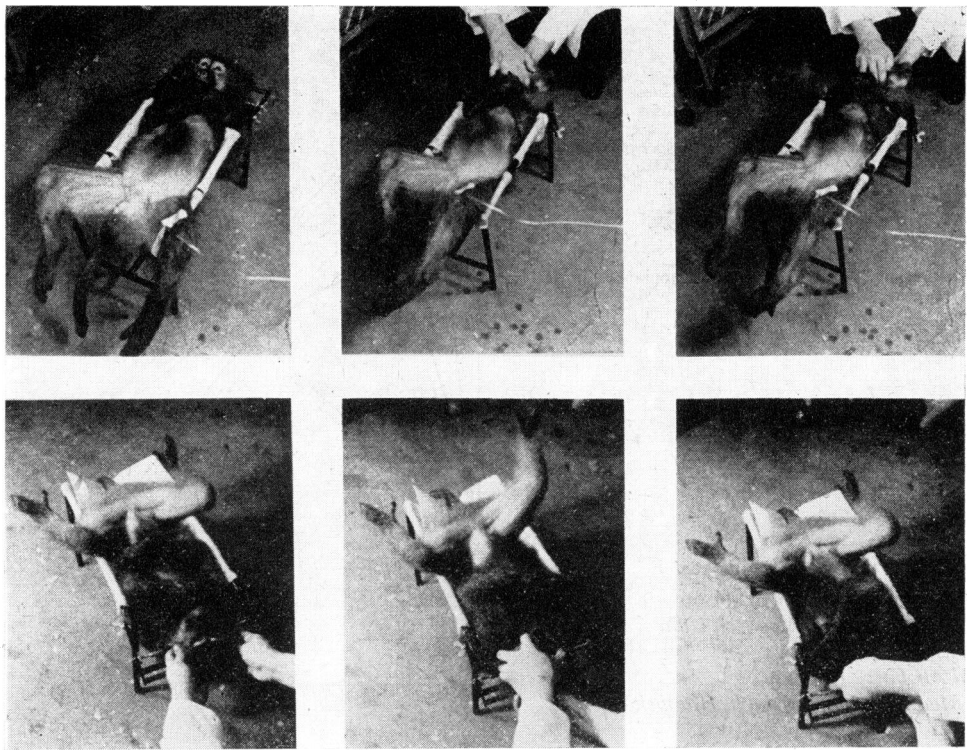

Fig 2 *Decorticate posture adopted by the two baboons. The tonic neck reflexes are being elicited in each animal. (Reproduced from Nicholson & Ernsting, 1967, by kind permission)*

with immediate survival but there was evidence of permanent brain damage in some of the survivors.

In two animals with permanent brain damage their immediate behaviour was similar. They remained motionless and adopted two characteristic postures depending on the manner in which they were supported. In the lateral position the undermost extremities were extended and the uppermost extremities were flexed while on their backs the fore limbs assumed a flexed posture and the legs tended to take up a semi-flexed position. Tonic neck reflexes were present but were easier to elicit in one animal. The resting posture was changed by rotation of the neck to the left. The right arm and leg (skull limbs) flexed slightly and the left leg (jaw limb) extended. On slight rotation of the neck to the right from this posture the right leg extended (Fig 2). The animal remained in this decorticate state, but after a couple of days the neck reflexes became more difficult to elicit and it died on the fifth day.

A similar response was seen in the other baboon but the animal made a surprising though partial recovery. During the remaining period of its survival it had considerable difficulty in maintaining the upright position and tended to support itself in the corner of the cage or by resting on the fore limbs. Unsteadiness of gait and ungainly postures were apparent. The animal clutched the side of the cage with widely spaced legs and on leaving the cage lost balance and toppled over. The ability to perform skilled movements with the hands had been lost and food was picked up with the mouth. The right arm would move across the front of the left arm and both limbs would move toward the mouth in an attempt to secure the food between the teeth. Frequently the co-ordination of these movements was defective (Fig 3).

Neuropathological examination (Brierley & Nicholson 1969*a*) of these two animals revealed extensive neocortical damage centred on the boundary zones between the territories supplied by the major cerebral arteries. Damage was symmetrical and decreased forwards from the occipital lobes. It took the form of laminar necrosis particularly in the 3rd, 5th and 6th layers. In one animal the cortical damage died out 2–3 cm from the frontal poles, but in the other it extended almost to the poles. Ischæmic necrosis was also seen in the hippocampi and in the caudate and lentiform nuclei of both animals and in the

Fig 3 *Resting position and feeding habits of the baboon which made a partial recovery from the decorticate posture. (Reproduced from Nicholson & Ernsting, 1967, by kind permission)*

thalamus of one. There was a slight loss of Purkinje cells in the cerebellum of each animal and marked cell loss in the substantia nigra, inferior colliculi and vestibular nuclei of one. A slight diffuse pallor of myelin staining in the central and subcortical white matter of both animals was present and there was a gliomesodermal reaction in all areas of grey matter damage.

More recent studies have revealed that the exposure of monkeys (*Macaca mulatta*) in air to a steady environmental pressure equivalent to 37 500 ft also leads to brain damage. These decompressions were of considerable interest because they have been used to define the respiratory and cardiovascular circumstances which can lead to brain damage (Ernsting & Nicholson 1971).

The behavioural effects of brain damage of a limited nature have also been explored (Nicholson *et al.* 1970, Blagbrough *et al.* 1973). Visual discrimination and tests of spatial and object alternation are not impaired for the days immediately after decompression but within a few days behavioural disturbances are apparent. These changes depend on damage in regions of the brain which are believed to be essential for the particular task and, although neuropathological evidence of brain damage must be present, factors other than neuronal loss – possibly œdema or epileptiform activity – would appear to contribute to the impairment. Recovery of function proceeds in the presence of limited but permanent brain damage.

From a practical point of view it has been necessary to define decompression profiles in supersonic transports which would be compatible with survival and which would avoid brain damage. These studies have been based on data which are applicable to forthcoming supersonic transports. Neurological, electrocortical and neuropathological studies (Brierley & Nicholson 1969*b*) established that, during decompressions with a peak altitude not exceeding 36 000 ft and of duration not exceeding 8 minutes above 10 000 ft, the spontaneous electrical activity of the brain would be maintained (though dominated by slow waves) and there would be no subsequent evidence of brain damage. Such a profile would not be fatal and would not lead to permanent brain damage in healthy passengers even breathing air provided pulmonary ventilation was maintained. The expected decompression profiles are little different from those which may follow decompressions in present-day subsonic transports and this is due to the rapid emergency descent of over 7000 ft per minute which can be effected by supersonic aircraft (Fig 1).

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Studies on the Nervous System of a Pilot During the Approach and Landing of a Transport Aircraft

Analyses of aircraft accidents frequently include pilot error as, at least, a contributory factor, but it is usually very difficult to unravel the circumstances which were believed to have led to impaired performance. In many cases the pilot was involved in a high workload situation, such as the approach to an airfield, and he could have been distracted from the essentials of the task or have experienced difficulty in processing all the information which the situation demanded. Much more needs to be known about the function of the nervous system under such stressful conditions.

Recent studies related to this problem have used a Boeing 707 of the British Overseas Airways Corporation (Nicholson *et al.* 1970). In each flight the individual factors which influenced overall workload were assessed by the pilot (i.e. technical serviceability of the aircraft, efficiency of the crew, availability of navigational aids, meteorological conditions, physical features of the airport and efficiency of air traffic control procedures) and the activity of the nervous system was followed by recording the electrocardiogram and finger tremor. The mean RR interval of the electrocardiogram was calculated for periods of specific interest such as around touch down and finger tremor was recorded before the flight and as soon as possible after touch down when the aircraft was brought to a temporary halt at the end of the runway.

In landings which did not involve undue workload on the part of the pilot the mean RR interval during the initial approach was usually around 500 milliseconds (heart rate around 120/min).